The rhomboidei major and minor: These muscles as such were absent, their place being occupied by white connective tissue whose fibers took the same general direction as those of the missing muscles would have done.

The serratus magnus: This muscle was continued upward and inward beyond the vertebral border of the scapula, and interlaced with the rhomboid fibers, making a strong network in which two layers of rather stringy muscle fibers lay.

Upon cutting the scapula away from the trunk two slips from the serrarus magnus were encountered, superior to the main muscle and its attachment to the vertebral border of the scapula. One of these inserted into the superior angle of the scapula, and had its origin from the scalenus anticus, medius, and posticus muscles. The other arose from the first rib and the scalenus posticus and inserted into the vertebral border of the scapula.

Anomalous muscles: Immediately posterior to the levator anguli scapulæ was a small slip of muscle which had its origin from the cervical aponeurosis opposite to the fourth cervical vertebra. Its insertion was very close indeed to that of the levator anguli scapulæ and the fibers ran in the same direction, but the two were not continuous. It was supplied by a twig from the fifth cervical nerve, which sent branches into the levator also, so that a fair conclusion would seem to be that it was an accessory levator anguli scapulæ, having its origin from the cervical aponeurosis.

From this description it will be seen that the elevated right scapula could have been movable only to a very slight extent. Most of the muscular attachments would tend to pull the bone upward or at least help to retain it in its high position, and a glance at the scapula or the bony arch will show that there was great muscular strain on these parts.

SOME REMARKS ON THE TREATMENT OF AMEBIC DYSENTERY.

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Within the last two or three years the scientific view-point concerning amebic dysentery and its treatment has undergone a decided change. While there may be present concomitant pathological conditions requiring appropriate attention, it is recognized that the crux of the whole therapeutic problem in this form of dysentery is to destroy the amebæ, and later to heal the ulcerated intestines and cure other complications.

For the destruction of the amebæ the treatment has practically narrowed down to two methods of medication: (1) internal and (2) hypodermic, and in the practice of the writer one method of intestinal irrigation, which is apparently a most useful adjunct. Granted this, the physician may be spared trying the numerous remedies which are recommended, many of which are utterly without merit. These three methods are the internal administration of ipecac, the hypodermic administration of emetin hydrochloride, and the injection into the large intestine of kerosene oil.

INTERNAL ADMINISTRATION OF IPECAC. For many years ipecac has been held in high esteem in East India and the neighboring Oriental countries, and while in some quarters it is unfavorably regarded it has the endorsement of Manson in England, Siler in this country, and others of authority. We have employed it in seven cases, with quite satisfactory results.

In giving large doses of ipecac, several details must be strictly observed or the stomach will not retain it, and consequent failure will ensue.

First of all the drug should be administered only in pill form, and these pills should be coated about a sixteenth or an eighth of an inch with phenyl salicylate (salol), for this coating does not readily dissolve in the stomach before it is absorbed and the violent emetic effect is avoided. The pills should generally contain 5 grains each of the ipecac.

If desired, castor oil may be given ten to twelve hours before the ipecac is to be taken, and a convenient method is as follows: No food is allowed for six hours and no water for three hours. Twenty drops of tincture of opium should be administered thirty minutes before the ipecac pills. The patient should be kept on the back and the pills swallowed with as little fluid as possible. After taking them he should not be allowed to move or speak or expectorate, and the nurse should keep a cold cloth over his throat and frequently bathe his face with cold water. The head should be kept low and both physical and psychic quiet enjoined. After an hour has elapsed he may gently move on his right side to facilitate the passage of the pills through the pylorus, but he should be allowed no water for three hours at least, and then sparingly. By observing these precautions the pills are generally retained; but should they be rejected the same procedure may be repeated the following day or night. After six or seven hours a sufficiency of water is admissible or any light nourishment desired.

The dose of ipecac should be about 40 or 50 grains the first night (eight or ten pills) and reduced 10 grains each night until only 10 grains are given. Following this somewhat rigorous treatment it is well to continue this 10-grain dose every alternate night for ten days to two weeks.

Given this way ipecac appears almost as a specific, and under its influence the stools become soft and mushy, but soon lose their dysenteric character, and may assume a normal form in a few days. The amebæ generally disappear almost at once, seldom reappearing, though occasionally they do, and then the treatment has to be repeated in its entirety.

HYPODERMIC ADMINISTRATION OF EMETIN. The *modus operandi* of ipecac in its amebacide properties was not well understood until Vedder, in March, 1911, working in Manila, published his findings that a fluidextract of ipecac would kill amebæ in cultures in dilutions as high as 1 to 200,000. The next year Rogers, in Calcutta, found that emetin hydrochloride killed amebæ in stools in dilutions of 1 to 100,000 and began to use this salt hypodermically in cases of amebic dysentery. Since then this preparation has been extensively employed, and favorable reports have come from Asia, Europe, North and South America, and "the isles of the sea."

The dosage recommended by Rogers, and followed by most observers, is from $\frac{1}{3}$ to 1 grain, injected twice a day, daily, or every second day for a week or ten days only.

At present, as contended by Allan, the evidence in regard to the permanence of cure by emetin is extremely meager. In the cases reported by Rogers (32) the active amebæ disappeared promptly after doses of $\frac{1}{3}$ to 1 grain; one case is reported well five months later, and in several of these cases which later came to autospy from other causes no amebæ were found in sections of the gut. There has been no report on the later history of the remainder of this series of cases, but Rogers thinks they are permanently cured.

Many other apparently permanent cures are reported, including either the amelioration or cure of liver abscesses, but there seems rather a paucity of reports following these cases any considerable length of time. Allan, of Charlotte, reported two cases which promptly cleared up and as promptly relapsed.

Most observers admit that the emetin always kills the active amebæ but not the cysts, the latter being responsible for the relapses. Baermann and Heinemann have detailed in full the treatment of twenty-two cases in Sumatra, furnishing some valuable data. They point out that emetin seems to exert but little effect on cysts and that there is considerable variation in the different preparations of emetin now on the market, as cases which proved refractory to one brand sometimes recovered promptly when emetin from a different manufacturer was employed. One important conclusion at which they have arrived is that while emetin is strongly amebacidal there are certain races of emetin-fast amebæ.

The writer has used this salt in four cases, three of which are still well at one year, eleven months, and ten months respectively. The fourth case suffered a relapse after an apparently normal condition of the bowels for two months, but he fell into other hands, so that details and subsequent symptoms are not available.

At present emetin hydrochloride is the salt mostly used. The size of the dose varies from $\frac{1}{4}$ to 4 grains, and it is also being given by the mouth and rectum—in rare instances intravenously. It is being given daily in single or several doses, on consecutive days, on alternate days, and at irregular intervals. Most observers discontinue it after the clinical symptoms subside or the amebæ disappear from the stools. Allan, a careful and safe clinician, recommends maximum doses—that is, 2 or 3 grains—instead of small doses. As no one has reported bad results except a transient nausea, and as small doses do not satisfactorily eradicate the amebæ, the conclusion of Allan seems sound and practicable.

The employment of emetin should be reinforced by rest in bed, appropriate diet, and other measures indicated for ulcerated intestines, perhaps abscessed liver, and a depleted body. As yet we do not know the best method as to dosage, frequence, or duration of treatment in the administration of emetin, but the mass of clinical reports now being poured into our literature will soon definitely settle these questions.

INJECTION OF KEROSENE OIL. This procedure is of recent date and has not come into general use, but is a valuable aid in the management of amebic dysentery. For an adult about 1 pint of pure oil should be employed, and with the patient in the knee-chest position, it should be injected into the bowel with a colon tube. He should keep this position, in an exaggerated manner if possible, for several minutes so that the oil will gravitate well into the colon. This should be retained for thirty or forty minutes and then be permitted to escape, though if all is not at once evacuated no alarm need be felt. R. T. Dorsey reports one case where, through a misunderstanding of orders, the oil was allowed to remain in the bowel over two hours. No ill effects occurred. We have not in any instance depended upon the oil alone in amebic dysentery, but in the stubborn diarrhea accompanying pellagra we have in a number of instances noted marked improvement following this procedure alone after various astringents had failed to afford relief.

This injection may be repeated daily for three or four days, and then occasionally as required. It will be found, however, that not many will be needed, for unless the case is extremely far advanced improvement will promptly set in.

Amebic dysentery, like duodenal ulcer, is probably much more prevalent than has been supposed, and its frequency has been shown by more thorough diagnostic methods. The only way a positive diagnosis of amebiasis can be made is by the microscope, and the finding of the protozoa in the stools. In regions where the disease is endemic and a microscope is not available one can reasonably infer its presence by the most prominent symptoms, namely, abdominal soreness, which is increased on pressure and extends along the course of the colon, especially when there is maximum intensity over the cecum and ascending colon. I believe with Musgrave, that the presence in the stools of any form of ameba in warm or tropical regions should be considered diagnostic for purposes of treatment, whether or not active symptoms are present.

Thus by carefully examining every suspected case of possible amebic infection and by early and energetic treatment along the lines herein considered this otherwise serious disease can in nearly every instance be promptly and permanently relieved.

THE DIAGNOSIS OF TUBERCULOSIS IN EARLY LIFE.[1]

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This paper has to do with the chronic form of pulmonary tuberculosis in children, and does not consider the acute manifestation of the disease, such as general miliary tuberculosis, tuberculous meningitis, or the acute bronchopneumonic form which complicates or follows some one of the acute infectious diseases; nor does it consider the surgical aspects of the disease. There are two reasons for this: In the first place, children suffering from the disease in any of the forms above mentioned usually are taken to the general hospitals and are rarely seen in a dispensary devoted exclusively to tuberculous patients, and in the second place, while individual cases may present difficulties in making a diagnosis, the clinical picture in the majority of cases of this type is usually readily recognized.

Excluding the types just mentioned the recognition of tuberculosis in children is either extremely easy or most difficult, the determination of a positive or negative finding depending almost entirely on the attitude of the particular observer. Thus, in some instances, all children having a tuberculous parent have been considered as being likewise tuberculous. In others the occurrence of a positive tuberculin reaction or the presence of palpable lymph nodes in the neck have been considered as sufficient evidence to designate the child as having tuberculosis.

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[1] Read before the Philadelphia Pediatric Society, May 12, 1914.